4:45

767-4 Elevated Defibrillation Threshold When Right-Sided Venous Access is Used for Nonthoracotomy ICD Lead Implantation

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Because the defibrillation threshold (DFT) is related to the vector of energy delivery and amount of myocardium included in the shock field, we hypothesized that implantable defibrillator (ICD) leads implanted from the right side would yield higher DFTs than those implanted from the left. The database of the Endotak lead trial using a biphasic ICD (P2, CPI) was used to determine if the DFT depended on the site of venous access. The lead is a tripolar endocardial lead capable of bipolar sensing and pacing, and defibrillation using the lead alone (LA). The most commonly used models were the 0062 and 0064 leads (13 and 16 cm between the two defibrillation electrodes, respectively). The lead may be also linked to a subcutaneous/submuscular patch electrode (SQ/SM) using a Y-connector. Lead placement data were available from 595 (97.5%) of the 610 patients (pts) in the trial, and of those 345 (58%) had the DFT, defined as the lowest energy that was successful in terminating ventricular fibrillation, determined by a step-down protocol. There were 274 males and 71 females, with a mean age of 61 ± 13 years, and LV ejection fraction (LVEF) 0.33 ± 0.13. The cardiac disease was ischemic in 247 pts (72%), nonischemic cardiomyopathy in 74 pts (21%), and other etiologies in the rest. The endocardial lead was implanted from the left in 324 pts (93.9%) and the right in 21 pts (6.1%). There were no differences between the two groups with respect to gender, age, LVEF, or underlying cardiac disease. The mean DFTs (joules) were:

	LA ± SQ/SM	LA (all)	LA 0062	LA 0064
N	345	230	32	198
Left approach	9.9 ± 4.8	10.1 ± 5.0	10.6 ± 6.1	10.1 ± 4.8
Right approach	14.0 ± 7.3	14.6 ± 6.6	15.0 ± 0.0	14.5 ± 7.3
P value	0.02	<0.01	0.13	0.04

Conclusion: Nonthoracotomy ICD leads implanted from the right yield higher biphasic DFTs than those implanted from the left confirming the important influence that lead position and vector have on the DFT.

5:00

767-5 Differential Effects of Lidocaine on Defibrillation Threshold with Monophasic and Biphasic Waveforms

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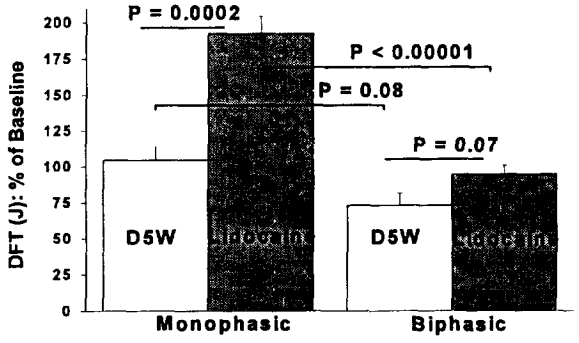
Antiarrhythmic drugs can interfere with electrical defibrillation using monophasic non-sequential shocks (MS). It is unknown if these drugs affect the efficacy of biphasic defibrillation shocks (BS). Since MS and BS waveforms can exhibit disparate effects on myocardial excitability and refractoriness (properties which may influence defibrillation efficacy), it is possible that antiarrhythmic drugs, such as lidocaine (L), will affect defibrillation threshold (DFT) of one waveform differently than another. We studied 25 pentobarbital anesthetized farm swine who had endocardial pacing and monophasic action potential catheters placed into the right ventricle. Pacing was used to induced sustained ventricular fibrillation (5–8 s) which was followed by defibrillation via epicardial electrodes. Each pig was assigned to one of four treatment groups; 1) MS + D5W (n = 7), 2) MS + L (n = 7) 3) BS + D5W (n = 5), or 4) BS + L (n = 7). DFTs were measured at baseline (Base) and subsequently during treatment (D5W or L). DFT values (joules) predicting 50% success at baseline were: MS + D5W = 7.34 ± 1.59, MS + L = 7.11 ± 1.5, BS + D5W = 5.28 ± 1.55 and BS + L = 3.86 ± 1.2. The figure depicts



treatment DFT values as percent of baseline. In the MS groups, the change in DFT from Base to L was 92 ± 28% vs the change from Base to D5W of 0.6 ± 29% ($p < 0.0001$). In the BS groups, however, the change in DFT from Base to L was similar to the change from Base to D5W (−6.7 ± 15 vs −29 ± 17%, $p = 0.08$). Compared to D5W, L increased DFT in the biphasic group by a magnitude that was $\frac{1}{4}$ that seen in the monophasic group. *Clinical Implications:* The effect of antiarrhythmic drugs on DFT (especially increased DFT values) may be less of a concern when using implantable devices employing biphasic waveforms.

5:15

767-6 Effect of Lead Polarity on the Defibrillation Threshold of Pectorally Implanted Cardioverter Defibrillators

Edward T. Keelan, Jasbir Sra, Kathi Axtell, Cheryl Maglio, Vinay K. Bahl, Mohammad R. Jazayeri, Anwer Dhala, Zalmen Blanck, Sanjay Deshpande, Michael Biehl, Masood Akhtar *Sinai Samaritan/St. Luke's Medical Center, Milwaukee, WI*

The effect of the polarity of the initial phase of a biphasic shock waveform on the defibrillation threshold (DFT) of cardioverter-defibrillators (ICDs) is not known. We tested this in two investigational pectorally implanted biphasic ICDs — Medtronic Models 7219C and 7219D PCD Jewel devices — in 22 consecutive patients (Pts). The 7219C has an "active can" and requires a single right ventricular (RV) lead while the 7219D requires RV and superior vena cava leads ± subcutaneous patch(es). The 7219C was implanted in 10 Pts and the 7219D in 12 Pts. Polarities were tested in random order. The results were:

	RV+*	RV−*	p value
7219C DFT	6.6 ± 3.1 J	10.8 ± 5.5J	0.007
Impedance	59.3 ± 8.8 Ω	60.3 ± 7.0 Ω	0.33
7219D DFT	12.0 ± 4.0 J	16.3 ± 7.3 J	0.07
Impedance	50.5 ± 6.7 Ω	47.5 ± 8.9 Ω	0.23

*Refers to the polarity of the initial pulse deflection

Of the 10 Pts receiving a 7219C device, 7 had lower DFT with RV+ while in 3 lead polarity had no effect. Of the 12 Pts receiving a 7219D device, 7 had lower DFT with RV+, 2 had lower DFT with RV−, and in 3 lead polarity had no effect. Overall, with RV+ there was a 39% reduction in DFT for Model 7219C and a 31% reduction for Model 7219D. An implant criterion of DFT ≤24J was met in 21 Pts using either RV+ or RV−. In one Pt, however, the DFT for RV− was 34J and for RV+ was 12J.

Conclusion: In this series, the lowest DFT was achieved most often using the RV+ polarity. These results suggest that both RV lead polarities should be tested to achieve the lowest DFT in Pts receiving a pectorally implanted ICD.

768 Unstable Angina: The Plaque and the Artery

Tuesday, March 21, 1995, 4:00 p.m.–5:30 p.m.
Ernest N. Morial Convention Center, Room 6

4:00

768-1 Coronary Lesion Histology in Stable, Unstable and Evolving Angina Pectoris

James M. Wilson, Pavel Capek, H.A. McAllister, William K. Vaughn, James J. Ferguson, Fred J. Clubb Jr., L. Maximilian Buja, James T. Willerson. *St. Luke's Episcopal Hospital, Texas Heart Institute, Baylor College of Medicine, Houston, TX*

We performed a histologic analysis of coronary lesions obtained by directional atherectomy from 100 patients with stable, unstable and evolving angina pectoris. Histologic analysis was performed by two pathologists unaware of the clinical history or angiographic findings. This included notation of the presence or absence of various matrix components and quantitative scoring (0–3+) of cellular lesion components. Forty-seven patients (47%) had native coronary lesions. Twenty-five patients (25%) had restenosis after previous balloon angioplasty and twenty-eight (28%) were treated for primary saphenous vein graft disease. There were no differences in the average histologic appearance of atherosclerotic lesions resulting in stable, unstable and evolving angina pectoris. Saphenous vein graft lesions were typically rich in dense connective tissue and extracellular lipid and mononuclear infiltrate. Restenosis lesions were rich in acid mucopolysaccharide and vascular smooth muscle cells and revealed no histologic differences according to the clinical syndrome. Native coronary artery lesions, from patients with unstable angina, more often contained organizing thrombi (30% vs 0%, $p < 0.05$), cholesterol crystals (20% vs 0%, $p < 0.05$) and harbored a more intense vascular smooth muscle cell infiltrate (2.1 vs 1.4, $p < 0.05$) when compared to lesions from patients with stable angina pectoris. There was no difference

in the intensity of inflammatory or foam cell infiltration.

Conclusion: Vascular smooth muscle cells, appearing as intimal hyperplasia, are more prominent in lesions resulting in unstable angina. There is, however, no difference in the intensity of inflammatory or foam cell infiltrate. The histologic appearance of coronary lesions producing the syndrome of unstable angina pectoris is quite heterogenous. In order to establish the role of inflammation in the development of non-fatal acute coronary syndromes, improved sampling methods or a larger sample size will be required.

4:15

768-2 Non-culprit Lesions in Acute Myocardial Infarction: Implications for Multiple Plaque Rupture in Acute Coronary Syndromes

Hugo H. Castaño, Herman K. Gold, Tsunehiro Yasuda, Hiroyuki Matsuura, Robert D. Dinsmore, John B. Newell, Igor F. Palacios, Erling Falk, Pedro R. Moreno *Cardiac Unit, Massachusetts General Hospital, Harvard Medical School, Boston, MA*

Rupture of atherosclerotic plaques is the most important mechanism underlying acute coronary syndromes. Previous post-mortem studies have shown multiple plaque rupture sites during fatal coronary events. We tested the hypothesis that non-culprit lesions (NCL) decrease in severity after thrombolysis in patients with acute myocardial infarction (AMI). Quantitative coronary angiography was performed before and 9 ± 3 days after rTPA infusion for AMI in 61 consecutive patients (52 males, 9 females, mean age: 53 ± 9 yrs) with 118 NCL mean 1.9 ± 0.9 per patient.

	RD*	% Dst	% Area st	MLD*	P Area**	Length*
Pre	3.2 ± 0.9	52 ± 21	72 ± 17	1.58 ± 0.8	7.4 ± 6.1	8.3 ± 4.4
Post	3.2 ± 0.9	49 ± 22	69 ± 20	1.66 ± 0.9	7.0 ± 5.4	8.2 ± 4.4
p	NS	0.01	0.03	0.005	0.016	NS

R: reference; D: diameter; L: luminal; M: minimal; P: plaque; %: percent; st: stenosis; *: mm; **: mm^2

Conclusion: Non-culprit lesions decrease in severity after thrombolysis in acute myocardial infarction suggesting a reversible thrombotic phenomenon probably related to multiple coronary plaque rupture.

4:30

768-3 Yellow Plaques and Vessel Morphology Prior to Coronary Intervention. A Study Using Intracoronary Angioscopy

Jose Baptista, Pim de Feyter, Carlo di Mario, Jos R.T.C. Roelandt, Patrick W. Serruys *Thoraxcenter, Erasmus University, Rotterdam, NL*

Angioscopy provides a direct and chromatic visualization of the intimal surface of the coronary arteries, providing an opportunity to assess in-vivo the characteristics of the culprit lesion in stable and unstable syndromes. We performed angioscopy in 78 patients prior to coronary interventions. Twenty five had stable angina (SAP), 36 unstable angina (UAP) defined as classes IIB and IIIB of the Braunwald classification, and 17 patients had post-infarction angina (Braunwald IIIC) (POST-MI). The parameters evaluated in the stenotic segment were: 1) lumen shape: round or elliptical vs complex (COMPLEX). 2) intimal surface: ulcerated (ULC) vs non-ulcerated. 3) presence of yellow plaque (YEL). 4) presence of red thrombi (THR). A THR score was derived from THR morphology: single mural (1 point), multiple mural (2), protruding (3), occlusive (4), and then multiplied by the extent of segments with THR (proximal, mid and distal lesion). Angiography was used to classify lesions as unstable or stables according to a modified Ambrose classification. *Results:* An angiographic unstable lesion was present in 44%, 39% and 41% of the lesions, respectively in SAP, UAP and POST-MI with a predictive value for an angioscopic complex lesion of 0.63.

Angioscopy	SAP	UAP	POST-MI	p value
COMPLEX	5 (20%)*¥	14 (39%)*	11 (65%)¥	<0.05*¥
ULC	3 (12%)*¥	17 (47%)*	9 (53%)¥	<0.05*¥
Red Thrombus	4 (16%)*¥	25 (69%)*	14 (82%)¥	<0.05*¥
THR score	3.25 ± 2.22	4.96 ± 3.29	5.86 ± 4.66	ns
Yellow plaque	18 (72%)	22 (66%)	12 (71%)	ns

Thrombotic burden was higher, whenever a yellow plaque was present near a THR (THR score 3.08 ± 2.07, YEL present vs. 5.87 ± 3.93, no YEL, p < 0.05).

Conclusions: Vessel ulceration and thrombosis is frequently found in UAP and POST-MI, but is poorly predicted by angiography. Yellow plaques are present in the majority of the patients and when ruptured they are associated with a larger thrombus formation. This may partially explain the benefits of anti-lipid therapy.

4:45

768-4 What is Unique About the Infarct Related Artery and How Do These Features Influence Success During Direct PTCA for AMI

Sandeep Khurana, Theodore Schreiber, Debra Sachs, Donovan Bakalyar, Robert D. Safian, V. Reddy, William W. O'Neill, Cindy Grines. *William Beaumont Hospital, Royal Oak, MI*

In spite of frequent presence of "high risk" characteristics, infarct angioplasty is not fraught with adverse procedural complications. We analyzed angiographic morphology of 103 consecutive AMI pts undergoing direct PTCA for its influence on procedural success. Using multivariate analysis lesion length (p = 0.002) and infarct related artery calcification (p = 0.001) were the only features predicting success. TIMI 3 reflow was predicted only by the residual flow grade (TIMI flow + collateral flow) prior to PTCA and not by either components alone. AMI lesions characteristics were compared with those in 154 pts undergoing PTCA for chronic CAD or unstable angina as shown in the table.

LESION CHARACTERISTIC	Control pts Pre PTCA	AMI pts Pre PTCA	AMI pts Post PTCA
Irregular or ulcerated	2%	70%	
Clot	12%	69%**	8%‡‡
Haziness	12%	82%**	56%‡‡
% Stenosis Pre/Post	65%/30%	95%*	34%
Incidence of Dissection	30%		17%‡
Ave Dissection Length	4 mm		15 mm‡‡

*p < 0.05; **p < 0.01 Pre Vs Post AMI; ‡p < 0.05, ‡‡p < 0.01 AMI Pre Vs Post

Conclusions: Angiographic characteristics considered to be "high risk" for PTCA do not adversely affect procedural success in AMI, pointing to uniqueness of the underlying lesion. A paradoxically lower incidence but longer length of angiographic dissection after PTCA in AMI suggests that the nature of plaque rupture that leads to AMI is different from the injury induced by PTCA. Better luminal enlargement in spite of more severe initial stenosis is consistent with compression of soft plaque or thrombus as the principal mode of PTCA action in AMI. A significant transformation of morphology from high to low risk in a majority of pts undergoing direct PTCA for AMI may explain lowering of recurrent ischemia after direct PTCA. Unique angiographic characteristics can also predict TIMI 3 reflow as well as anatomic success for PTCA in AMI.

5:00

768-5 Evolution of Infarct-related Stenoses After Clinical Stabilization

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The evolution of infarct-related stenoses (IRS) was prospectively studied in 106 patients (pts) with myocardial infarction (MI) after clinical stabilization. All pts underwent diagnostic angiography (6 ± 5 months after MI, range 1 to 25) and were put on a routine waiting list for PTCA of the IRS. Coronary angiography was repeated (8 ± 4 months after first angiogram) immediately preceding PTCA (79 pts) or soon after the occurrence of coronary events (MI: 6 pts, unstable angina: 21 pts). IRS was identified and classified as "complex" (irregular borders, overhanging edges, or intracoronary thrombus) or "smooth". Stenosis progression, assessed by computerized angiography, was defined as >20% diameter reduction or new total occlusion and regression as >20% decrease in severity. At initial angiography, there were 279 stenoses (≥50:138, <50%:141). Of 106 IRS, 74 (70%) were complex and 32 (30%) were smooth. At restudy, 24 of 106 IRS had progressed compared with 9 of 173 non-IRS (23% vs. 5%, p = 0.001). Twenty-two of the 24 IRS and 3 of 9 of non-IRS that had progressed developed into total occlusion (92% vs. 33%, P = 0.01). Twenty-two of 74 complex IRS progressed, compared with only 2 of 32 smooth IRS (30% vs. 6%, p = 0.008). During follow up, coronary events occurred in 27 pts, of which 15 (56%) were associated with IRS progression. Remoulding of stenosis morphology (from complex to smooth) was found in 8 patients (resolution of thrombus or ulceration), of these 3 were associated with stenosis regression. Pts age, gender, coronary disease risk factors, number of diseased vessel, antiplatelet and antithrombotic therapy, interval between onset of MI and diagnostic angiogram, and interval between the 2 coronary angiograms were not associated with IRS progression. Changes of percent stenosis (%) and minimal stenosis diameter (mm) in the different subgroups were as following:

TUESDAY PM